Remarks

In response to the February 11, 2002 Notice, applicants submit a substitute paper copy of the Sequence Listing as **Exhibit D** and a computer readable form (CRF) of the Sequence Listing on diskette. Applicants request that the paper copy of the sequence listing attached hereto replace the previously submitted paper copy of the sequence listing. Moreover, applicants submit as **Exhibit E** a Statement In Accordance With 37 C.F.R. §1.821(f) certifying that the computer readable form and paper copy of the Sequence Listing are the same.

If a telephone interview would be of assistance in advancing prosecution of the subject application, applicants' undersigned attorney invites the Examiner to telephone him at the number provided below.

No fee is deemed necessary in connection with the filing of this Amendment and Communication. However, if any fee is required, authorization is hereby given to charge the amount of any such fee to Deposit Account No. 03-3125.

Respectfully submitted,

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I hereby certify that this correspondence is being deposited this date with the U.S. Postal Service with sufficient postage as first class mail in an envelope addressed to:
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CY1316 (*MATα gpa1Δ farΔ tbt1-1 fus1-HIS3 can1 ste14::trp1::LYS2 ste3Δlys2 ura3 leu2 trp1 his3*): The parent of all strains used in this study, was obtained by standard genetic techniques, with SY1390 (Stevenson et al. (1992) *Genes Dev.* 6:1293-1304) (provided by G. Sprague), and SM1188 (Sapperstein et al. (1994) *Mol. Cell. Biol.* 14:1438-1449) (provided by S. Michaelis) serving as the original sources of the *fus1-HIS3* and *ste14* alleles, respectively. Unless otherwise indicated, all genomic disruptions were made with the *URA3* gene, followed by selection on 5'-fluoroorotic acid (Boeke et al. (1987) *Methods. Enz.* 154:164-195). Gα genomic integrations were made at the *GPA1* locus and verified by Southern, Gα expression, and phenotypic analysis. Plasmid pR15 (Beals et al. (1987) *Proc. Natl. Acad. Sci. USA* 84:7886-7890), carrying the coding region of human Gαi2. Plasmid CP1127, carrying the promoter sequences and first 41 amino acid codons of *GPA1*, was prepared by ligation of a sequence encompassing nucleotides –200 to +100 of *GPA1* (where translational start is +1) to pRS405 (Sikorski and Hieter (1989) *Genetics* 122:19). Plasmid CP1183, carrying the $GPA1_{(1-41)}$-Gαi2 chimera sequence, was made by PCR amplification of the Gαi2 coding region encompassing amino acids 36 to its stop codon at position 357 using the oligo pair SEQ ID NO:73 and SEQ ID NO:5 and using plasmid pR15 as template. The amplified product was digested with *Sac*I and *Sal*I, then ligated into *Sac*I/*Sal*I digested CP1127. A glycine to alanine alteration at codon 204 of Gαi2 in CP1183 was introduced using Stratagene's QuickChange kit and mutagenic oligos SEQ ID NO:6 and SEQ ID NO:7 creating plasmid CP5533. Sequences encoding β-galactosidase (*lacZ*) were introduced downstream of the *fus1* promoter on plasmid pRS424 (Sikorski and Hieter (1989) *Genetics* 122:19) to create CP1584. Plasmid pSM187, with a 4.3 kb DNA fragment carrying the *STE14* gene flanked by *Bam*HI sites, was kindly provided by S. Michaelis. This *Bam*HI fragment was inserted into *Bam*HI digested and shrimp alkaline phosphatase treated pRS415 and pRS414 (Sikorski and Hieter (1989) *Genetics* 122:19) to create, respectively, plasmids CP5108 and CP5336.

The yeast phosphoglycerate kinase (*PGK1*) promoter sequence was amplified from yeast genomic DNA using the oligonucleotide pair SEQ ID NOs: 26 and 27, digested with *Bgl*II and *Nco*I, and ligated into *Esp*3I/*Nco*I digested Yep51Nco (Broach

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